Acute exacerbation of COPD (AECOPD)

August 19, 2024 by **Josh Farkas**

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rapid reference

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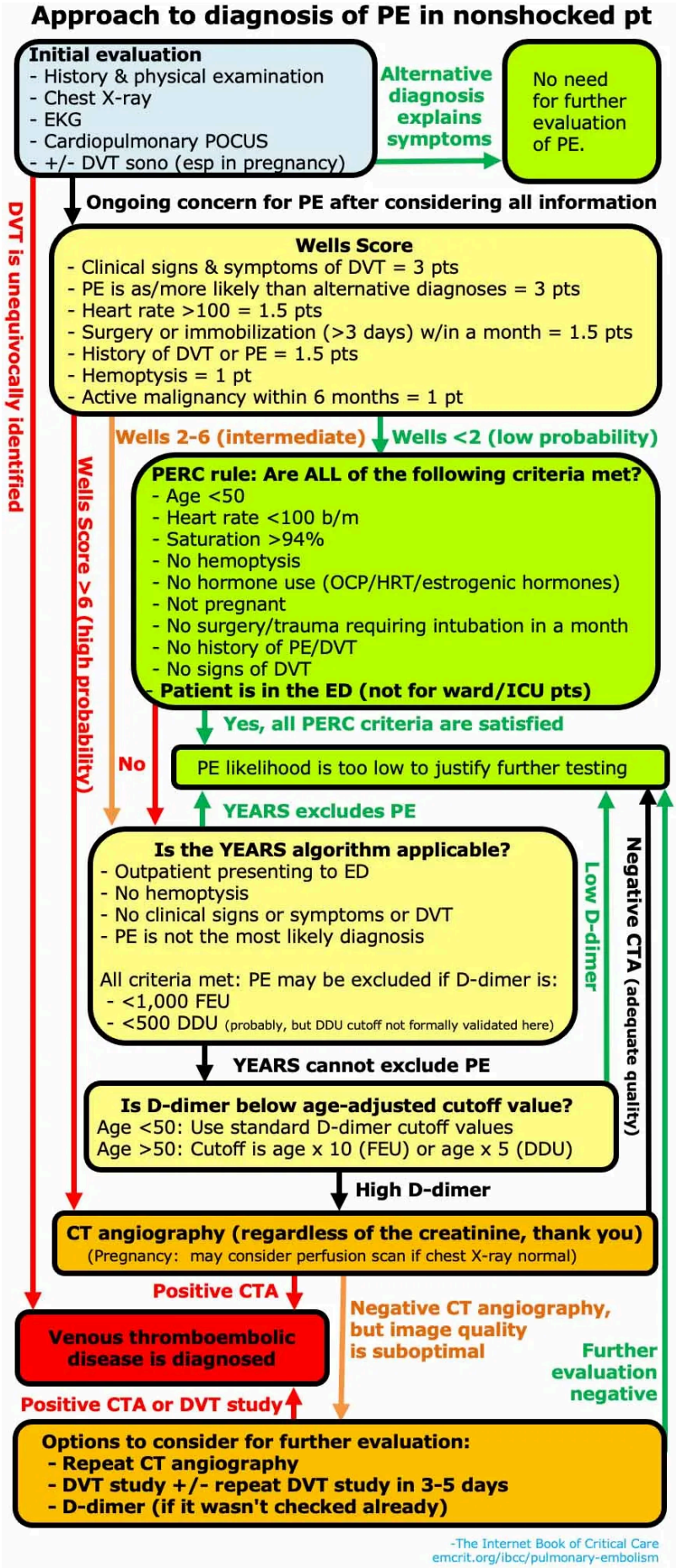
confirm diagnosis

- Review history, laboratory studies, and chest radiographs.
- Further tests as needed (e.g., PE workup if atypical symptoms).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steroid

- Usually, start with IV methylprednisolone (e.g., 125 mg IV).
- Rapidly de-escalate to lower doses (e.g., prednisone 50 mg qd).

bronchodilators

- Hold all home bronchodilators.
- Albuterol/ipratropium nebulized q6hr scheduled.
- Albuterol nebulized q2hr PRN.

antibiotic

- Narrow-spectrum is adequate (typically azithromycin 💉 (https://emcrit.org/ibcc/antibiotics/#macrolides_(azithromycin,_clarithromycin)) or doxycycline 💉 (https://emcrit.org/ibcc/antibiotics/#doxycycline)).
- Try to choose something the patient wasn't recently exposed to.

respiratory support in non-intubated patient

- BiPAP is the first line:
  - Remember to titrate settings to optimize the level of support.
  - Trial sedation if unable to tolerate (e.g., dexmedetomidine). 📖 (#intolerance)
  - Contraindicated if nausea/vomiting.
- HFNC is the second line.
- Intubation only if clinically necessary.
- Monitoring:
  - BiPAP: Follow tidal volume & minute ventilation.
  - Respiratory rate: <10 suggests hypoventilation, >30 suggests high work of breathing that may be unsustainable.
  - Oxygen saturation: Target 88-92%.
  - Mental status/CO2:
    - If the patient is arousable and mentating, follow mental status.
    - For patients who are sedated, follow ABG/VBG.
  - Gestalt: Probably most important. Look at the patient. Discuss with the nurse and respiratory therapist. Follow over time.

management of the intubated COPD patient

- Ventilator settings:
  - Avoid tachypnea & autoPEEP.
  - Target respiratory acidosis (e.g., pH 7.25-7.35), but greater degrees of permissive hypercapnia may be beneficial to avoid barotrauma.
- Start enteral nutrition early (patients often have baseline malnutrition).

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evaluation

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history and physical including:

- History:
  - ? Cough.
  - ? Change in sputum volume/purulence.
  - ? Dyspnea severity and trajectory.
  - ? Fevers, night sweats, rigors.
  - ? Hemoptysis.
  - ? Chest discomfort.
  - ? Weight gain, ankle edema.
  - ? Asymmetric leg swelling and/or pain.
- Examination:
  - Vital signs.
  - Auscultation of the lungs and neck.
  - Evaluation for peripheral edema or asymmetric leg swelling.

radiological studies

- POCUS of heart and lungs.
- Chest radiograph.
- ECG.
- **CT angiography**: for more complex patients, primarily if there is ongoing concern regarding the possibility of pulmonary embolism.

laboratory studies

- CBC, electrolytes.
- D-dimer if PE is a clinical consideration (and arguably in all COPD patients).
- BNP or pro-BNP if heart failure is a consideration (BNP <100 pg/mL or pro-BNP <300 pg/mL may largely exclude heart failure; elevated values are less specific). (36701677 (https://pubmed.ncbi.nlm.nih.gov/36701677/))
- Viral PCR (e.g., COVID, influenza PRN).
- ABG/VBG?
  - Generally unhelpful and unnecessary.
  - Compared to ABG, VBG is generally fine and more humane. 📖 (https://emcrit.org/ibcc/vbg/)
  - ABG/VBG *doesn't* generally help diagnose AECOPD or differentiate it from other diagnoses. (21663600 (https://pubmed.ncbi.nlm.nih.gov/21663600/))
  - ABG/VBG is primarily helpful in a patient with altered mental status to evaluate for hypercapnic encephalopathy.
- ⚠️ Sputum gram stain and culture are *not* helpful (unless there are concerns regarding the possibility of pneumonia or bronchiectasis).

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common differential diagnostic challenges in AECOPD

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Patients with a history of "COPD" frequently present to the hospital with dyspnea. Most of them have AECOPD, but some don't. The following section focuses on diagnoses that are harder to sort out from AECOPD (for example, it doesn't include pneumothorax or pleural effusion – since these entities are more easily ascertained with a basic evaluation).

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[1] pneumonia

- Pneumonia may be extremely challenging to differentiate from AECOPD (*both* AECOPD and pneumonia may cause fever, chills, purulent sputum, and leukocytosis).

- The key differentiating factor is the presence or absence of a radiographic opacity. Unfortunately, chest radiography isn't 100% sensitive for pneumonia. In cases which are hard to tease apart, options include:
  - Chest CT scan (although it is generally not worth getting a scan solely for this reason).
  - Procalcitonin (if <0.5 ng/ml, this argues strongly against typical bacterial pneumonia).
- AECOPD and pneumonia often occur together ("pneumonic AECOPD" – the pneumonia is *causing* a COPD exacerbation). Treatment of pneumonic AECOPD consists of treating *both* pneumonia and COPD.
- A common approach for a patient with COPD and possible pneumonia is the following:
  - (1) Start on antibiotic coverage for pneumonia (e.g., ceftriaxone plus azithromycin) and check a procalcitonin.
  - (2) If procalcitonin is low (<0.5 ng/ml), this argues against typical bacterial pneumonia. Ceftriaxone can be discontinued, while azithromycin is continued to treat COPD.
  - (3) If procalcitonin is elevated, then continue combination antibiotic therapy for pneumonia (along with full-bore COPD therapy as well – the presence of PNA doesn't exclude concomitant COPD).

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[2] PE (pulmonary embolism)

epidemiology

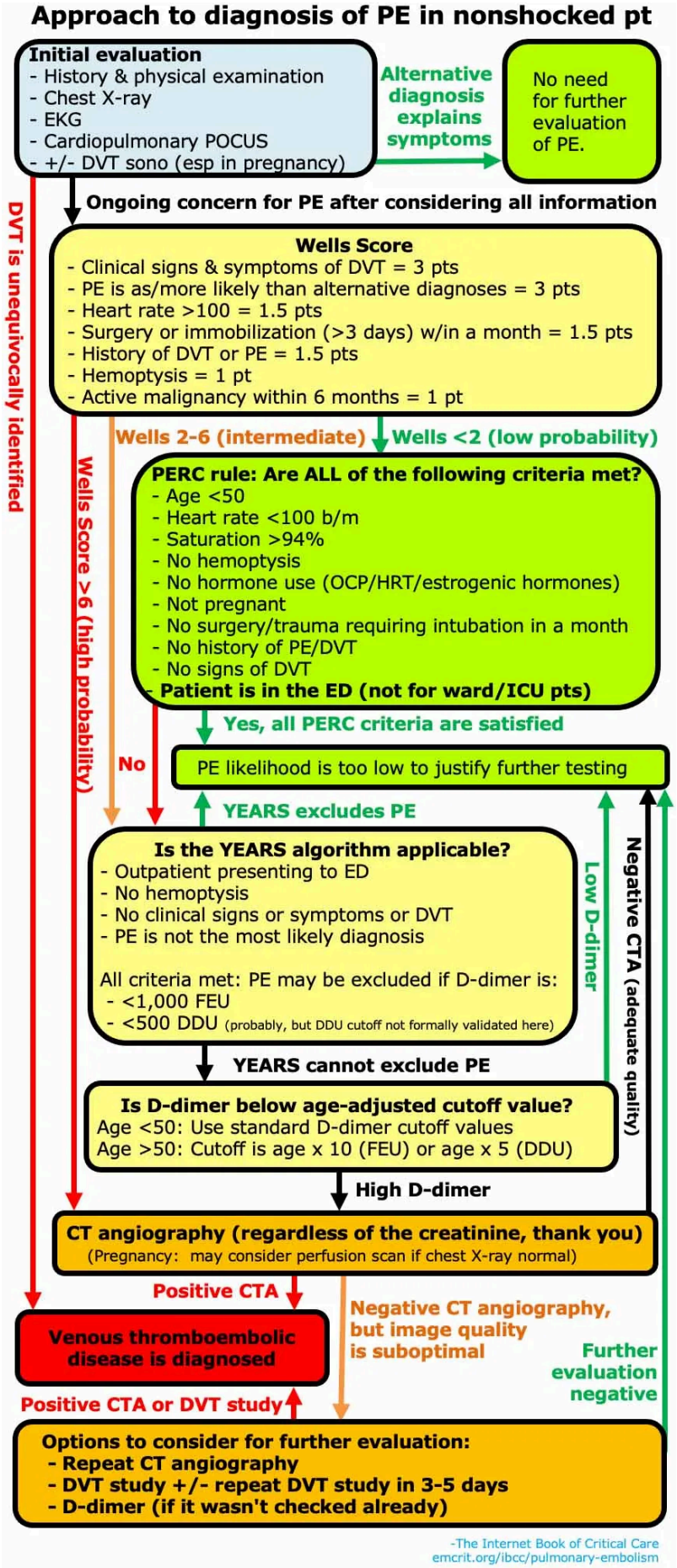
- PE is found in 12% of patients who are diagnosed with AECOPD. The mortality of such patients is elevated, suggesting potential harm due to PE (rather than simply incidentally discovered PE). (34879475 (https://pubmed.ncbi.nlm.nih.gov/34879475/))
- This is notable because, on average, a patient with AECOPD has a PE frequency that is above the test threshold for PE (12% >> 2%). Even among patients in whom PE is not suspected, the rate of PE remains significant (4%). (36701677 (https://pubmed.ncbi.nlm.nih.gov/36701677/)) This implies that we should be more diligent about considering and screening for PE in this patient population.

features that should increase consideration for PE

- [1] Any atypical COPD exacerbation should raise suspicion for PE (e.g., lack of purulent sputum, fever, chills, different in quality from patient's prior COPD exacerbations).
- [2] Risk factors for PE, including:
  - Prior venous thromboembolic disease (DVT/PE).
  - Recent surgery or trauma (especially orthopedic).
  - Hospitalization for CHF, MI, or AF within three months.
  - Malignancy.
  - Older age.
- [3] History or physical examination suggestive of DVT.

approach to PE diagnosis

- The approach to PE diagnosis is shown below and discussed further here: 📖 (https://emcrit.org/ibcc/pulmonary-embolism/) .
- D-dimer is often mildly elevated in patients with AECOPD. However, a negative D-dimer may often be sufficient to exclude PE (using the algorithm below).
- When in doubt, obtaining a CT angiogram to exclude PE is often reasonable:
  - Contrast dye doesn't cause renal failure. 📖 (https://emcrit.org/ibcc/contrast/)
  - Radiation-induced malignancy is less of an issue for patients old enough to have COPD.
  - Many of these patients may be eligible for chest CT scans to screen for lung cancer, as roughly outlined below: 📖 (https://emcrit.org/ibcc/lung-cancer/#lung_cancer_screening)
    - 50-80 years old.
    - ≧20-pack-year smoking history.
    - Currently smoking, or quit within the past 15 years.
    - Does not have a health problem that substantially limits life expectancy.
    - Is willing and able to undergo curative lung surgery.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[3] heart failure

- Key findings to support a diagnosis of heart failure may include:
  - Bilateral B-lines on ultrasonography.
  - Signs of congestion on the chest radiograph.
- Patients with severe COPD often have some right ventricular dysfunction and systemic congestion. Consequently, AECOPD is often associated with peripheral edema and elevated BNP levels.
- BiPAP application may cause rapid resolution of B-lines on POCUS, making it difficult to determine whether there was heart failure in retrospect. This may be one situation where adding on a BNP or pro-BNP level to admission labs could be reasonable.

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[4] upper airway obstruction

- There are numerous potential causes of upper airway obstruction (e.g., tumor, angioedema, epiglottitis, vocal cord dysfunction).
- Key findings that reveal upper airway obstruction may include:
  - The physical exam revealing stridor that is grossly audible without a stethoscope.

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[5] AEOHS (acute exacerbation of obesity hypoventilation syndrome)

- Patients with AEOHS present with acute-on-chronic hypercapnic respiratory failure that mimics COPD and is frequently misdiagnosed as COPD.
- Clues to the diagnosis of AEOHS:
  - Elevated body mass index.
  - Laboratory data of chronic hypercapnia (e.g., chronically elevated serum bicarbonate levels).
  - Absence of other features of AECOPD, e.g.:
    - No wheezing.
    - No sputum production.

- Many patients with COPD may have a COPD/OHS overlap syndrome. Such patients may require treatment for both disease processes.
- (OHS is discussed further here: 📖 (https://emcrit.org/ibcc/sleep/#obesity_hypoventilation_syndrome_(OHS)))

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[6] sedating medications

- Patients with severe COPD may have a relatively weak respiratory drive, rendering them increasingly sensitive to the respiratory suppressive effects of sedatives and opioids.
- Among COPD patients who are on chronic opioids, it can be extremely difficult to sort out whether hypercapnia is a medication side-effect or is due to underlying COPD.

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[7] asthma

- Trying to sort out asthma from COPD can be very challenging (discussed further here: 📖 (https://emcrit.org/ibcc/copd/#differentiation_of_asthma_vs._COPD)).
- It may be impossible to differentiate asthma from COPD.
- The treatment of COPD and asthma are very similar. When in doubt, design a treatment plan that adequately treats both processes. This will generally look like a therapeutic plan to treat asthma, plus the addition of antibiotics.

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[8] bronchiectasis & BCOS (bronchiectasis-COPD overlap syndrome)

- COPD and bronchiectasis may have similar presentations (e.g., chronic cough and mucus production). Additionally, ~30% of patients with COPD have BCOS. 📖 (https://emcrit.org/ibcc/bronchiectasis/#bronchiectasis_and_COPD_overlap_syndrome_(BCOS))
- History and thoracic CT scans may help sort out these possibilities (including *archival* CT scans).
- Some patients may have elements of both AECOPD and bronchiectasis, which may be treated with management strategies for both diseases (e.g., AECOPD therapies with

aggressive airway clearance and perhaps more aggressive antibiotics).

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basic treatments

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steroid

- There is no precise evidence on how to dose steroid for COPD patients in the ICU.
- A reasonable *initial approach* might be:
  - Start with 125 mg IV methylprednisolone in the emergency department.
  - If the patient remains on the verge of requiring intubation, then continue methylprednisolone 125 mg IV daily for 1-2 days.
- Once patients are stable/improving:
  - Reduce steroid to 30-60 mg/day prednisone for 5-10 days. (Fishman 2023)
  - 50 mg prednisone x5 days is usually sufficient.
- **Nebulized budesonide**:
  - Nebulized budesonide probably shouldn't be utilized for severe COPD exacerbation in the ICU. However, some evidence does support the use of nebulized budesonide for mild exacerbations of COPD. (11874817 (https://pubmed.ncbi.nlm.nih.gov/11874817/) , 32161453 (https://pubmed.ncbi.nlm.nih.gov/32161453/) , 17251232 (https://pubmed.ncbi.nlm.nih.gov/17251232/))
  - Nebulized budesonide could be a rational therapy for patients with mild COPD exacerbation plus contraindications to systemic steroids (e.g., brittle diabetes, history of severe steroid-induced psychosis).
  - RCTs have validated using *enormous doses* of nebulized budesonide (1.5-2 mg q6hr). These doses may be logsitically difficult to deliver in some hospitals.

bronchodilators

- [1] Hold all home inhalers. Acutely ill patients are usually too breathless to take their home medications properly (metered-dose inhalers, etc.).
- [2] The following regimen of bronchodilators is adequate:

    - Albuterol plus ipratropium nebulized q4hr or q6hr *scheduled*.
    - Albuterol nebulized Q2hr PRN.
- For patients on BiPAP or HFNC, bronchodilators should be nebulized and administered in-line through the device (without removing the patient from support).

antibiotics

- Patients sick enough to be in the ICU due to COPD should receive antibiotics (regardless of whether there is an opacity on the chest radiograph). (23235687 (https://pubmed.ncbi.nlm.nih.gov/23235687/))
- Patients with COPD have airways that *chronically* grow a variety of organisms. Antibiotic therapy aims to suppress this bacterial growth, not to completely sterilize the patient's lungs (which is impossible). Therefore, narrow-spectrum antibiotics are fine.
    - Avoid getting sputum cultures and ignore them if they have been obtained (these patients will grow weird stuff in their sputum chronically; there is no need to cover every single organism). (20066545 (https://pubmed.ncbi.nlm.nih.gov/20066545/))
- Good choices:
    - Azithromycin 💉 (https://emcrit.org/ibcc/antibiotics/#macrolides_(azithromycin,_clarithromycin)) is generally first-line if the patient hasn't been exposed to it recently. (Best available evidence indicates that azithromycin doesn't increase QTc significantly or causes Torsade de Pointes. 🌊 (https://emcrit.org/pulmcrit/myth-busting-azithromycin-does-not-cause-torsade-de-pointes-or-increase-mortality/))
    - Doxycycline 💉 (https://emcrit.org/ibcc/antibiotics/#doxycycline) is also an excellent choice (given its high tolerability and low risk of C. Difficile).
- For patients with BCOS 📖 (https://emcrit.org/ibcc/bronchiectasis/#bronchiectasis_and_COPD_overlap_syndrome_(BCOS)) (bronchiectasis-COPD overlap syndrome), using broader antibiotics similar to a treatment strategy for bronchiectasis could be considered. However, there is no prospective evidence regarding this.

avoid excess oxygen

- Excess oxygen may cause diffuse pulmonary vasodilation, which disrupts ventilation-perfusion matching and thereby increases PaCO2.(23106947 (https://pubmed.ncbi.nlm.nih.gov/23106947/))

- Titrate inhaled oxygen to target an oxygen saturation of 88-92% (with 85-95% being OK).

roflumilast

- Roflumilast is an oral phosphodiesterase-4 inhibitor that increases intracellular levels of cAMP (similar to theophylline).
- There is no evidence to support the *initiation* of roflumilast during an acute exacerbation of COPD.
- For patients already on roflumilast, continuing this therapy may be rational (if it is on the hospital's formulary).
- (Further discussion of roflumilast here: 📖 (https://emcrit.org/ibcc/copd/#roflumilast))

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noninvasive ventilatory strategies

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indications for immediate intubation

- Immediate intubation is generally the wrong move. Intubation can often be avoided by strategically using various medications and noninvasive modalities.
- Indications for immediate intubation may include:
  - Multiorgan failure (e.g., COPD plus cardiogenic/septic shock)
  - A patient who is genuinely not protecting the airway (e.g., gurgling secretions in the upper airway)
  - Respiratory/cardiac arrest
- When in doubt about intubation, a reasonable approach is often to prepare for intubation while simultaneously placing the patient on BiPAP.
  - If the patient improves, that's great; you can avoid intubation.
  - BiPAP will still optimize their physiology before intubation if the patient doesn't improve.

BiPAP is the first-line noninvasive strategy

- BiPAP is supported by a robust evidence base for treating COPD. It has been proven to reduce death (relative risk 0.4), reduce intubation (relative risk 0.4), and reduce treatment complications (relative risk 0.3). This is impressive evidence that argues strongly that the patient should be given a real college try on BiPAP whenever possible.
- Indications for BiPAP?
  - Substantial respiratory distress or tachypnea (respiratory rate >~30/min).
  - Somnolence due to hypercapnic encephalopathy, as a result of COPD exacerbation.
- Contraindications to BiPAP
  - Need for immediate intubation (see above).
  - Vomiting or increased risk of vomiting (e.g., bowel obstruction).
  - Copious secretions, difficulty with secretion management.
- BiPAP settings
  - Pressure: Start at 10cm iPAP/5 cm ePAP. If tolerated, may up-titrate as needed to ~18 cm iPAP/8 cm ePAP. Titrate the driving pressure (iPAP-ePAP) to achieve an adequate tidal volume. Using a slightly elevated ePAP to cancel out autoPEEP (e.g., 8 cm) may facilitate triggering the BiPAP more easily and reduce the work of breathing.
  - Monitor tidal volume & minute ventilation on the BiPAP monitor.
  - Really low tidal volumes (e.g., <300-400 ml) and low minute ventilation (e.g., <5-6 L/min) suggest inadequate ventilation. In this situation, try up-titrating the pressures and widening the driving pressure (with a rough maximum support level of around ~20cm iPAP/5 cm ePAP).

what if the patient can't tolerate the BiPAP mask?

- Don't just assume that the patient needs to be intubated. The first step here is often to try some sort of **sedation**. If that fails, then the patient may be trialed on **HFNC**.
- **Dexmedetomidine**
  - Excellent anxiolytic to help patients tolerate the mask and rest while on BiPAP. The combination of BiPAP plus dexmedetomidine is termed "BiPAPidex." This is a powerful approach, especially for anxious patients with flash AECOPD (see figure above).
  - The strength of dexmedetomidine is that it doesn't suppress the respiratory drive, and it's titratable, making it the safest sedative.

    - The weakness of dexmedetomidine is that it can take a little while to work. Boluses of dexmedetomidine can cause hemodynamic instability, so a reasonable approach may be to start the infusion at a high rate (1-1.4 mcg/kg/hr) and then titrate down as the patient becomes sleepy. This will take ~30-60 min to really work.
- **IV haloperidol, droperidol, or olanzapine** are other options that may calm patients without suppressing respiratory drive or causing delirium. These agents have the added benefit of functioning as antiemetics.
- **Benzodiazepines** have mixed results here: occasionally, they work, but they often cause the patient to get more confused/agitated. For patients who are on benzodiazepines chronically and respond well to this class of medication, administration of benzodiazepines makes sense.
- **Fentanyl**: For patients with severe tachypnea and air hunger, small divided doses of fentanyl can be used to help them decrease their respiratory rate sufficiently to give them time to exhale appropriately. This must be done with very careful monitoring of minute ventilation & respiratory rate by someone with extensive experience treating respiratory failure. IV fentanyl is preferable because it works rapidly, so you can titrate it meticulously to hit a sweet spot where the patient is breathing at a rate of ~12-24 breaths/minute (fast enough to stay alive but slow enough to allow sufficient time to exhale).

high-flow nasal cannula (HFNC)

- HFNC helps COPD patients primarily by reducing their anatomic dead space, improving ventilation, and reducing the work of breathing ("blowing off CO2" – more on the chapter on ***HFNC***).
- There isn't much evidence to support the use of HFNC in COPD (unlike BiPAP, which is supported by robust evidence). Thus, HFNC is currently a second-line therapy here.
- HFNC may be useful in the following situations:
    - Patients who are unable to tolerate BiPAP.
    - Patients in whom BiPAP is contraindicated (e.g. due to vomiting), but who aren't sick enough to require intubation.
- Titration of settings:
    - **The flow rate should be maximized** to the highest level the patient will tolerate (ideally at least 50-60 liters/minute flow).
    - FiO2 should be adjusted to target a saturation of 88-92% (accepting sats of 85-95%).

monitoring on BiPAP/HFNC

- Key parameters
  - **Oxygen saturation**: Target saturation 88-92%, tolerate 85-95%. Excessive oxygen may impair VQ matching and thereby impair CO2 clearance.
  - **FiO2 requirement**: COPD generally impairs CO2 clearance but shouldn't cause profound hypoxemia. If the patient has escalating oxygen requirements, this suggests something else is going on (e.g., pneumonia, mucus plugging, pulmonary embolism) – you need to investigate this further.
  - **Respiratory rate**. Significant tachypnea (e.g.,>~25-30 b/m) is a bad sign that the patient may eventually tire out. Ideally, HFNC/BiPAP should cause a drop in respiratory rate.
  - **Work of breathing**: Look for deterioration (e.g., retractions, abdominal paradoxical breathing, tripoding).
  - **BiPAP monitor**: BiPAP allows you to monitor tidal volume & minute ventilation as described above. Note that a mask leak may cause these measurements to be imprecise.
- ABG/VBG *or* mental status
  - If the patient has an intact mental status, I don't think you need serial ABG/VBG values. If the patient is arousable and able to report how they are feeling, then just follow the clinical exam. Ideally, the patient will report that they are feeling better. If the patient starts getting progressively more sleepy/confused, then you may be in trouble (check an ABG/VBG to exclude severe hypercapnia).
  - If the patient is sedated, then you do need to follow ABG/VBG values to make sure the patient isn't becoming dangerously hypercapnic (sedation prevents you from using mental status to exclude severe hypercapnia). While either ABG or VBG is fine, serial VBG monitoring using a peripheral vascular catheter that allows blood withdrawal is usually the most humane approach. 📖 (https://emcrit.org/ibcc/vbg/)

indications for delayed intubation

- *Delayed intubation* is defined here as a patient who is stabilized on BiPAP or HFNC but subsequently is intubated a couple of hours later (usually due to failure to improve). This

is often a mistake because it represents a misunderstanding of the goals of noninvasive ventilation.

- Goals of BiPAP/HFNC in COPD:
  - (a) Maintain adequate oxygenation (>85-88%)
  - (b) Reduce the breathing work so the patient doesn't develop progressive diaphragmatic fatigue. This is probably the most important goal. Therapeutic targets here include improving tachypnea and the patient's subjective sense of breathlessness.
  - (c) Keep pCO2 low enough that the patient doesn't develop complete obtundation/coma. The goal is *not* to improve the ABG/VBG immediately. Ideally, the blood gas will improve, but this may take a while (several hours).
- Key points:
  - The decision to intubate is ultimately a *clinical decision*. If the patient seems stable or improving but the blood gas remains unchanged, then it's often best to continue BiPAP/HFNC with careful monitoring.
  - Serial ABG or VBG values will vary randomly by as much as ~0.03 differences in pH and ~5 mm differences in pCO2.(19091262 (https://pubmed.ncbi.nlm.nih.gov/19091262/) , 8020270 (https://pubmed.ncbi.nlm.nih.gov/8020270/) , 6407807 (https://pubmed.ncbi.nlm.nih.gov/6407807/) , 1583545 (https://pubmed.ncbi.nlm.nih.gov/1583545/)) So, if the pCO2 increases slightly, that doesn't necessarily indicate that the patient is worsening – it may simply be noise due to repeated laboratory measurements.
- In summary, intubation is indicated for clinical deterioration. However, if the patient is overall stable, then it's often best to continue noninvasive therapy with careful monitoring (with the ability to immediately intubate if warranted). It may take some hours for the bronchi to open up and clinical resolution to occur. Wait for it.

how long should BiPAP/HFNC support be continued?

- For most patients, ~12-24 hours of support may be reasonable.
- Avoid premature discontinuation of support. Diaphragmatic fatigue and bronchoconstriction take time to resolve. Even if the patient looks beautiful after 1-2 hours on BiPAP, it's often a mistake to discontinue it prematurely (assuming that the patient truly needed BiPAP initially).

    - One potential exception is a patient with pure flash-COPD exacerbation (see figure above). This refers specifically to a patient who was doing perfectly fine, then suddenly developed anxiety/tachypnea and fell apart. If the patient was really doing great before this episode, they might require only transient BiPAP support to stabilize them and return to their baseline.
- Don't keep patients on BiPAP for too long. Over time, BiPAP can cause ulceration of the nose. It's probably a bad idea to leave a patient on continuous BiPAP for >48 hours. If the patient is unable to be freed from BiPAP after 48 hours of intensive therapy (e.g., even unable to tolerate HFNC), then you probably need to consider intubation.
- For patients who are very tenuous and require a prolonged duration of support, the following strategies may be considered:
    - HFNC can be continued indefinitely because this allows for adequate nutrition.
    - Many patients can be weaned from BiPAP to a combination of nocturnal BiPAP plus HFNC during the day. Over time, as they recover, they can be transitioned to nocturnal BiPAP plus a standard low-flow nasal cannula during the day.

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intubation & ventilator management

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indications for intubation

- These are explored in more detail above. To summarize:
- (a) Indications for immediate intubation
    - Multiorgan failure (e.g., COPD plus cardiogenic/septic shock).
    - The patient is really not protecting their airway (e.g., gurgling secretions).
    - Respiratory/cardiac arrest.
- (b) Indications for delayed intubation
    - The patient is clinically deteriorating despite optimized BiPAP/HFNC support.
    - The patient stabilizes on BiPAP but is completely BiPAP-dependent for >48 hours.

intubation procedure itself

- Consider using a relatively large-size ETT (e.g., 8.0 for larger people, 7.5 for smaller people). Using a small ETT may increase airway resistance, hindering your ability to ventilate.
- Resist the urge to aggressively bag patients following intubation. COPD patients may rapidly trap gas in their lungs (due to impaired airflow), leading to pneumothorax or hypotension. Bag these patients *gently* and *slowly*.

ventilator settings

- Ventilating COPD patients is generally much easier than ventilating asthmatic patients, although both have airflow limitations.
    - COPD patients: Respiratory failure is usually due to a *combination* of diaphragmatic fatigue and bronchospasm. Once they are on the ventilator, diaphragmatic fatigue isn't a problem – so ventilation is relatively straightforward.
    - Asthmatic patients: Respiratory failure is due primarily to intense bronchospasm. The degree of bronchospasm is more severe, which can create major challenges in ventilator management.
- The primary concern with ventilation is autoPEEP
    - Patients have difficulty with expiration. If the tidal volume and/or respiratory rate are too high, this causes gas trapping inside the chest at end-expiration (autoPEEP).
    - AutoPEEP can be problematic because it can impair venous return to the heart (causing hypotension) and make it difficult for the patient to trigger the ventilator (leading to ventilator dyssynchrony).
    - AutoPEEP can be diagnosed by persistent expiratory flow at end-exhalation (airflow never goes to zero before the next breath).
    - The treatment for autoPEEP is to reduce the respiratory rate and/or tidal volume. Decreasing the respiratory rate is generally the most effective intervention. If the patient is over-breathing the ventilator, suppression of their respiratory rate may be necessary (e.g., with propofol or an opioid).
    - Increasing the set PEEP slightly (e.g., from 5 cm to 8 cm) may stent open airways during expiration and make it easier for patients with a little autoPEEP to trigger the ventilator.
- Reasonable starting settings:
    - Volume-cycled vent: Tidal volume 8 cc/kg, respiratory rate ~14 b/m, 5-8 cm PEEP.

- Pressure-cycled vent: Pressure 30 cm/8 cm, respiratory rate ~14 b/m.

target blood gas?

- Many COPD patients have chronic hypercapnic respiratory failure with chronic compensatory metabolic alkalosis. In this case, ventilation to a normal pCO2 (40mm) is problematic for two reasons:
  - (1) Ventilation to a normal pCO2 will cause alkalemia (pH >7.45), which probably isn't awesome.
  - (2) The kidney will respond to alkalemia over time by excreting bicarbonate until the serum bicarbonate level is ~24 mEq/L. This will cause problems with trying to get the patient off the ventilator. Stripped of their chronic compensatory metabolic alkalosis, the patient needs to blow their pCO2 down to ~40 mm to achieve a normal pH. This will increase their work of breathing, making it harder for them to pass a spontaneous breathing trial or be liberated from the ventilator.
- The best approach is generally to target a pCO2 close to the patient's baseline value:
  - If you know the patient's baseline, you can use that.
  - In most cases, you won't know the patient's baseline. In this situation, targeting a slightly low pH (~7.25-7.35) will get you close to the patient's baseline pCO2. Mild acidemia will stimulate the kidney to retain bicarbonate, which keeps the patient near their baseline bicarbonate level (which will eventually facilitate extubation).
  - If there is difficulty achieving this pH, then lower pH may also be entirely acceptable (i.e., a strategy of *permissive hypercapnia*).
- Unfortunately, severe COPD is one situation where end-tidal CO2 may be misleading.

weaning off ventilation

- Timing:
  - It's generally a reasonable idea to rest the patient on the ventilator for at least ~24 hours to allow diaphragmatic rest. (Even if the patient looks terrific after a few hours on the ventilator, it's generally inadvisable to extubate at that point.)
  - After ~36-48 hours, bronchospasm and diaphragmatic fatigue should improve, so efforts to wean should be quite aggressive in that time frame.
- Prophylactic extubation to HFNC or BiPAP reduces the risk of extubation.
  - HFNC is more tolerable, potentially making it superior here.

- For patients with chronic hypercapnia, consider transitioning to chronic nocturnal BiPAP.

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key pathophysiologic concepts for management of COPD

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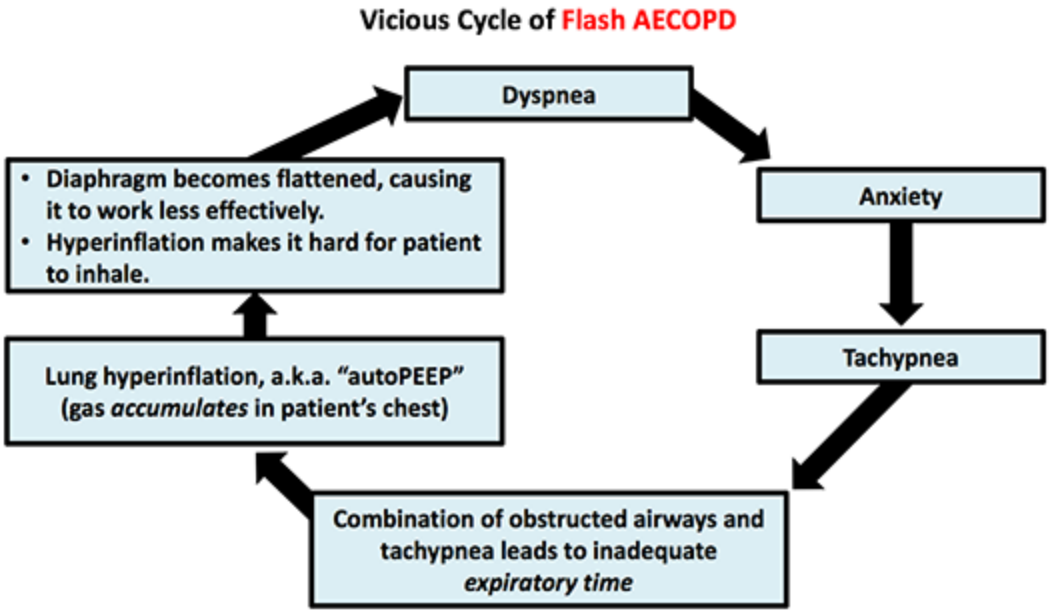
#1) concept of "Flash AECOPD"



Patients with COPD and anxiety may fall into a cycle shown above with progressive anxiety, tachypnea, dyspnea, and gas trapping. This may cause patients to deteriorate very rapidly but improve rapidly as well. A combination of BiPAP and anxiolytics may be very helpful in breaking patients out of an episode.

#2) concept of diaphragmatic rest

One of the central problems in AECOPD is the exhaustion of the diaphragm. After working hard for a prolonged period of time, the diaphragm becomes fatigued. Diaphragmatic fatigue may require 24-48 hours of rest to recover. Probably one of the critical roles of BiPAP or intubation is to rest the diaphragm. Take-home messages based on this concept:

- Even if the patient recovers well after a few hours on BiPAP, it may still be worthwhile to leave the BiPAP on longer (e.g. overnight) to rest the diaphragm.
- If a patient gets intubated for COPD, it is generally wise to leave them intubated for at least ~1 day before they are extubated (assuming that they genuinely required intubation in the first place).

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podcast

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questions & discussion

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To keep this page small and fast, questions & discussion about this post can be found on another page ***here*** (https://emcrit.org/pulmcrit/AECOPD/) .

- Over-use of antibiotics: Chasing sputum cultures with broad-spectrum antibiotics. As discussed above, COPD patients will always grow strange pathogens from their sputum, even when healthy (e.g., pseudomonas).
- Under-use of antibiotics: Failure to provide antibiotic therapy to a patient with severe AECOPD.
- Under-utilization of BiPAP: Even patients who look terrible (and may seem to require intubation) often improve rapidly on BiPAP.
- Inadequate sedation for BiPAP: BiPAP is proven to reduce mortality in COPD, so it's worth taking a little time and trying to sedate the patient so that they can tolerate it (e.g., with dexmedetomidine). Immediately concluding that an anxious patient "can't tolerate BiPAP" and proceeding to intubation often isn't in the patient's best interest.

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- 🌊 = Link to FOAMed site with related information.
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